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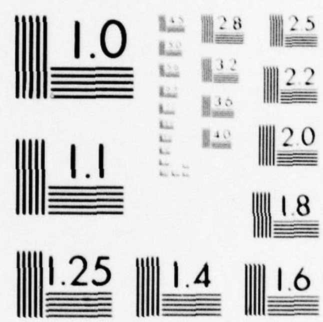

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Effects of Hyperbaric Oxygenation on Acute Alcohol Intoxication

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20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

This equipment loan contract was entered into on November 1, 1974 and terminated on August 31, 1979. Title to the equipment involved, a Scientific Research Instruments Corporation MS-8 Medical Mass Spectrometer, U. S. Government I.D. #4172, had been transferred to St. Luke's Hospital at the termination of the contract.

Previously, under another contract from the Office of Naval Research, we had carried out studies of nitrogen and helium elimination from man during

decompression. The results of these experiments were published in Undersea Biomedical Research (Vol. 2 (4):277-297, 1975).

The initial studies carried out under this equipment loan contract were to repeat the published studies but this time with the subjects completely immersed in water. Mark S. Low, a physiology graduate student at the Medical College of Wisconsin, was the principle investigator for this work. Mr. Low's experiments were completed by 1977 and the results published in a Master's Thesis (Low, M.S. (1977), Master's Thesis, Department of Physiology, Medical College of Wisconsin, (Medical College of Wisconsin Library). This thesis has been previously submitted to the Office of Naval Research.

Following the completion of Mr. Low's studies, the mass spectrometer was used intermittently to determine the efficiency in oxygen delivery of different kinds of oxygen masks used in this facility. It has also been used on a regular basis to analyze breathing gas mixtures used in the treatment of decompression sickness and air embolism. These gas mixtures are prepared in this laboratory and analyzed against calibrated standards with the mass spectrometer before use. The instrument also played an integral part in analyzing normoxic breathing gases for an experiment to determine the effect of breathing hyperbaric oxygen on ethanol metabolism. In the ethanol experiments, six subjects were given sufficient alcohol to produce legal intoxication and then subjected to breathing hyperbaric oxygen at 2 atmospheres absolute or 11.5% oxygen at the same pressure. The metabolic rates or elimination curves for ethanol were found to be identical for both test situations. However, simple hydrostatic pressure seemed to increase the metabolism of ethanol as under both hyperbaric conditions (normoxic and hyperoxic) ethanol was metabolized faster than when breathing air at atmospheric pressure. This study was carried out in cooperation with De Paul Rehabilitation Hospital. The results of this study at present are still in press, but the manuscript is appended.

At the present time, funding is being sought for the production of new decompression tables for compressed air tunnel workers in the United States through the National Institute of Occupational Safety and Health. At this time, this proposal had been approved on the basis of scientific merit and given priority, but we do not have official notification of actual funding. In these proposed studies, the mass spectrometer would be used again to perform nitrogen wash-out studies during and after decompression on present decompression tables for tunnel workers and new experimental tables. Nitrogen washout would be one way the new tables could be compared to the old in terms of their decompression efficiency. The nitrogen wash-out studies are planned in conjunction with ultra-sonic imaging of static tissue bubbles in these tests. The mass spectrometer has become an integral part of the investigative armamentarium of this laboratory and new uses for it are anticipated in the future.

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Principle Investigator

Effects of Hyperbaric Oxygenation on Acute Alcohol Intoxication[1]

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A great deal of documentation is available regarding the acute and chronic negative effects of alcohol ingestion on cognitive, intellectual, perceptual, motor, and memory processes, as well as other behavioral and physiological functions (e.g., Mello and Mendelson, 1970; Goldstein and Neuringer, 1976), particularly as these abilities and processes relate to driving and traffic safety. And for at least forty years scientists have been studying the rate at which alcohol is absorbed into the blood stream, distributed throughout the body, and finally metabolized and excreted (Haggard, Greenberg, and Lolli, 1940; Mallach, Olderhausen, and Springer, 1972). There has been a continuing interest in the relationship between blood alcohol concentration (BAC) and the ability to perform a variety of complex and simple tasks (Mello and Mendelson, 1970), and a pursuit of the chimerical "sobering up" agent (from black coffee to cold showers) that will accelerate the metabolism and excretion of alcohol and rapidly return the intoxicated individual to his or her sober state of functioning.

Reports of success in reduction and/or reversal of acute and chronic effects of alcohol intoxication have appeared in the Soviet literature (Tishchenko, 1976; Spivak, 1977). "Discontinuation of psychomotor excitation and general hyperkinesia" and

"a more rapid restitution of consciousness and a relatively short development of soporous and comatose conditions... and a disappearance of psychotic disturbances" were observed following hyperbaric oxygen therapy (i.e., the delivery of higher than normal levels of oxygen under conditions of increased atmospheric pressure). Relatedly, groups of elderly patients with cognitive, intellectual, and memory deficits attributable to oxygen deficits associated with cerebral ateriosclerosis, showed significant reversals of those deficits when treated with a series of hyperbaric oxygen therapy sessions (Jacobs, Winter, Alvis, and Small, 1969).

The similarities in appearance between the deficits associated with aging and those caused by alcohol ingestion, and the at least partial success in reducing and/or reversing those effects through use of hyperbaric oxygen therapy, stimulated us to attempt a pilot study on the efficacy of relatively brief hyperbaric oxygenation on ameliorating the effects of acute ethanol intoxication among nonalcoholic social drinkers. Although other researchers had used prolonged and/or frequent exposures to hyperbaric oxygenation (e.g., Jacobs et al. used twice-daily sessions for fifteen days), we saw no point in extending the treatment period beyond the length of time that would have been required for natural unaided detoxication to occur.

Consequently, we hypothesized that under oxygen-enriched hyperbaric conditions (1) there would be an acceleration of alcohol

metabolism as reflected in a more-rapid decline of BAC during a two-hour period, and (2) at the end of the first hour the intoxicated social drinkers would show less of a performance deficit on relevant measures than would be shown under normal conditions or non-oxygen-enriched hyperbaric conditions. We also speculated that (3) in the absence of alcohol intoxication, performance might even be enhanced by an oxygen-enriched hyperbaric environment in comparison to performance under normal conditions.

Method

Subjects. Participants in this study were six healthy adult male students from the University of Wisconsin (Milwaukee). All were experienced social drinkers without history of alcohol or other drug problems. None had ever been exposed to hyperbaric conditions, and none were trained or experienced scuba-divers. Their average age was 29.6 years (standard deviation = ± 13.21 years), and none of the participants were using medications or drugs while the experiment was in progress. Subjects were recruited by means of an advertisement in the University newspaper, and each was paid \$20.00 per session plus a \$40.00 bonus after completion of all sessions.

Apparatus. A large walk-in double-lock hyperbaric chamber (26'L x 9'W x 7'H), capable of accommodating six adults seated and three experimenters/technicians and their equipment, operated

by the Department of Hyperbaric Medicine at St. Luke's Hospital, was used for all experimental and placebo (control) conditions. Within the chamber, Scott double-seal oronasal demand masks were used to deliver to each individual subject the appropriate gas misture (100% oxygen in the experimental condition, 11.5% oxygen and 88.5% nitrogen in the control condition). Blood gas analysis was conducted by means of an Astrup Radiometer blood gas analyzer operated by a technician in the chamber. Blood alcohol concentrations were determined by means of a Smith and Wesson Breathalyzer model no. 90A, and an Abbot ABA-100 bichromatic analyzer.

Cognitive and motor performance measures included five consecutive 10-second left-handed and right-handed trials administered in standardized fashion using the finger oscillator of the Halstead-Reitan Neuropsychological Test Battery (Small, 1973), standardized trials on the forward and backward portions of the Digit Span subtest of the Wechsler Adult Intelligence Scale (Rapaport, Gill, and Schafer, 1968), standardized 90-second trials on the Digit Symbol subtest of the Wechsler (Rapaport et al., 1968), and a maximum of ten trials (discontinued if subjects attained the criterion of two consecutive errorless repetitions) on a serial learning task using the method of anticipation with 8-item lists of C-V-C nonsense syllables having a 20 percent association value (Hilgard, 1963).

Alcoholic beverages were prepared by mixing approximately 180 ml

of orange or grapefruit juice with 100 proof vodka in a ratio of 0.75 gm of ethanol (28.4 ml of beverage) per kg of subject's body weight (after Mallach et al., 1972). Placebo beverages were prepared by soaking a cottonball in vodka and wiping the rim of the glass with it, filling the glass with approximately 300 ml of juice, and floating approximately 10-15 drops of vodka on the surface of the juice.[2]

Magazines, books, and other reading materials were available to subjects at all times while they were in the hyperbaric chamber, except during cognitive and motor performance-testing periods.

Procedures. Prospective subjects were addressed at group sessions, were fully informed of all procedures,[3] risks, hazards, and benefits, and informed consent was obtained. Subjects were later seen individually for an interview to assess past and present alcohol and other drug use, claustrophobic experiences, and related matters, and were then provided a medical examination, chest x-ray, and a brief exposure to the hyperbaric chamber to to determine their ability to clear their ears and sinuses under twice normal atmospheric pressure (2 ATA, equivalent to 33 feet sea water depth). Baseline testing on all performance measures used during the experimental sessions was administered to sober subjects individually in advance of the first session.

Experimental sessions were scheduled approximately one week apart

except when school vacations intervened. Twenty-four hours prior to each session, subjects were telephoned and were reminded to abstain from the use of alcohol (beverage, mouthwash, etc.) and other drugs, and to have no food or drink (except water) for eight hours prior to the session. All sessions began at 8:00 a.m.

Using a 2 x 2 experimental design which varied the beverage served (alcohol or placebo) and the gases breathed (oxygen or oxygen and nitrogen), four conditions were established, and each condition was presented twice, for a total of eight sessions conducted under hyperbaric conditions of 2 ATA. Selection of conditions was randomized (see Table 1), but in the interest of economy and ease of operation, all subjects underwent the same conditions at the same time.

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insert Table 1 here

At the beginning of each session a Breathalyzer reading was taken to ascertain that each subject's blood alcohol concentration approximated a zero level (BAC $\leq$ 0.01). At ten-minute intervals thereafter, subjects began drinking their beverages on cue, and ingestion was timed for fifteen minutes, followed by a fifteen-minute waiting period, and a second BAC assessment. Subjects were then individually "locked" into the hyperbaric chamber,[4] where they remained seated for sixty minutes, breathing the assigned gas mixture via the individual masks. During this period subjects were allowed to read, play solitaire, etc.;

the breathing masks made conversation very difficult and talking was usually discontinued after initial attempts proved fruitless.

After sixty minutes an arterial blood sample was taken for blood gas analysis (sessions 2 and 4 only), and a venous sample was taken (all sessions) and refrigerated for later chemical analysis of BAC. Immediately thereafter, cognitive and motor performance measures were individually administered, beginning with the finger oscillator (approximately 150 seconds), and digit symbol (90 seconds). Breathing masks were then removed and subjects breathed chamber air briefly while the forward and backward digit span (approximately 180 seconds) and serial learning (approximately 400 seconds) tasks were administered.[5] Breathing masks were then returned and subjects remained seated quietly in the chamber for an additional fifty minutes.

At the end of that period, a second venous blood sample was taken and refrigerated for later BAC analysis, subjects were relieved of their breathing masks, individually escorted into the forward compartment of the chamber and, while breathing chamber air, were "locked" out (i.e., decompressed to normal atmospheric pressure over a 4-6 minute period) under supervision and were escorted to a lounge area. A third Breathalyzer reading was taken at this time, regardless of the type of beverage served. If alcohol had not been served, subjects were allowed to leave after an informal debriefing and short period of

observation. During sessions 2, 3, 4, and 7, however, subjects were required to remain in the lounge under supervision until subsequent Breathalyzer tests (administered at approximately 15-20 minute intervals) showed a BAC $\leq$ 0.05.

In addition to the sober baseline measures taken pre-experimentally (session 0), an intoxicated baseline session was conducted post-experimentally (session 9). This session, held in another section of the hospital away from all possible distractions, was identical in all important details to other sessions in which alcohol had been served, with the following three exceptions: breathing masks were not used and hyperbaric conditions were not in effect; blood samples were not taken; additional Breathalyzer readings were taken approximately every fifteen minutes after alcohol ingestion was completed.

Results

For all sessions in which alcohol was ingested, mean BAC as determined by Breathalyzer and/or blood-sample analysis indicated that the legal criterion of intoxication (BAC $\geq$ 0.10 mg%) had been fulfilled. Blood gas analyses, conducted during sessions 2 and 4 only, revealed mean arterial oxygen pressures in excess of 1200 millimeters of mercury (mm Hg), as opposed to normal levels of 90-100 mm Hg. Thus, the desired results--intoxication and hyperbaric oxygenation--were attained when the independent variables were appropriately manipulated.

For purposes of data analyses, pairs of sessions having identical independent-variable conditions were pooled, resulting in four groups of data (sessions 2 and 4, 3 and 7, 5 and 8, and 1 and 6); the sober and intoxicated baseline sessions--0 and 9--were treated as two independent groups of data. BAC results (expressed in mg %/dl as derived from breath and/or blood samples), finger-oscillator test results (scored as the mean number of taps per 10-second trial for the right and left hands separately), Digit Span test results (scored as the mean number of correct responses for forward series, backward series, and total), Digit Symbol test results (scored as mean number of correct responses per 90-second trial), and serial-learning test results (scored as mean percentage of correct responses per trial) are shown in Table 2.

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insert Table 2 here

When analysis of variance (ANOVA) was applied to these data, several expected (and many unexpected) results obtained. Looking first at the results of the finger oscillator tests, no differences were found for performance with the left hand under the six different experimental and control conditions. For right-handed (the preferred or dominant hand for all subjects) testing, however, performance was somewhat better ($p < .10 > .05$)[6] during the intoxicated baseline condition than during the alcohol + hyperbaric oxygen condition (sessions 2 and 4) and the alcohol + hyperbaric atmosphere condition

(sessions 3 and 7).

Performance on Digit Span forward was unaffected by manipulation of independent variables except in one comparison: The alcohol + hyperbaric oxygen condition resulted in somewhat better performance ($p < .10 > .05$) than did the intoxicated baseline condition. Digit Span backward was similarly unaffected in all but one comparison: Performance was significantly ($p < .05$) better under the alcohol baseline condition than under the alcohol + hyperbaric atmosphere condition (sessions 3 and 7). Digit Span total was unaffected in all cases, as was Digit Symbol.

Rote learning, as reflected in the acquisition of nonsense syllables, appears to be variously affected by experimental conditions when performance was analyzed on a trial-by-trial basis. On the first trial, performance was significantly ($p < .01$) better when intoxicated subjects breathed oxygen under hyperbaric conditions, as compared to performance of intoxicated subjects breathing a normoxyc gas mixture under hyperbaric conditions, or intoxicated subjects performing under normal (baseline) conditions. Performance on trial 1 was marginally ($p < .10 > .05$) better during the sober baseline session than during the intoxicated baseline session.

Several significant differences were found also on the third trial of the serial-learning task: Intoxicated baseline

performance was significantly ($p < .05$) better than performance of intoxicated subjects breathing a normoxyc gas mixture under hyperbaric conditions; performance was significantly ($p < .05$) better under hyperbaric oxygen conditions for sober subjects than under normal conditions for sober subjects (baseline session 0). When intoxicated subjects breathed a normoxyc gas mixture under hyperbaric conditions, their performance was significantly ($p < .01$) poorer than that of sober subjects breathing hyperbaric oxygen, and significantly ($p < .05$) poorer than that of sober subjects breathing normoxyc gases under hyperbaric conditions. Intoxicated subjects breathing hyperbaric oxygen performed significantly ($p < .05$) worse than sober subjects breathing hyperbaric oxygen, and sober subjects breathing hyperbaric oxygen performed significantly ($p < .01$) better than sober subjects breathing normoxyc gases under hyperbaric conditions.

Marginal differences ($p < .10 > .05$) were found for performance on trial 4 of the serial-learning task, with intoxicated baseline scores being better than scores for intoxicated subjects under conditions of hyperbaric oxygen and hyperbaric normoxyc gases. And on trial 10, sober baseline scores were marginally ($p < .10 > .05$) better than intoxicated baseline scores.

Statistical analysis of BAC values at ten-minute intervals (interpolated data derived by statistical regression) indicated no differences between hyperbaric oxygen _vs_. hyperbaric normoxyc

gases. However, the descending limb of the plotted BAC curve fell off much more rapidly for both of those groups than it did for intoxicated subjects under baseline conditions. This significant difference ($p < .01$) is illustrated in Figure 1 (and tables of BAC values are available from the first author upon written request).

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insert Figure 1 here

Discussion

Our initial hypothesis, that a single two-hour session of hyperbaric oxygen treatment would accelerate the descending limb of the BAC curve, was supported at an acceptable level of statistical significance ($p < .01$), as compared to the BACs under normal atmospheric and barometric conditions. It was also observed, however, that a 2 ATA barometric condition during which intoxicated subjects breathed a normoxyc (11.5%) gas mixture, also accelerated the decline of BACs to the same extent. It must be inferred, therefore, that the hyperbaric condition itself, rather than the oxygen enrichment, was the significant variable.

There are no equivalent human-subjects data available with which to compare the results of our pilot study, although several studies cited by Alkana and Syapin (1979) indicated that "increased partial pressures of oxygen at nonelevated

[atmospheric] pressures failed to enhance ethanol metabolism... or reduce ethanol's depressant effect..."(p. 168). Since we did not have a control condition in which intoxicated subjects breathed 100% oxygen under normal atmospheric pressure, our results are not directly comparable. Inferentially, however, since hyperbaric oxygenation did not accelerate the reduction of BAC beyond that induced by normoxyc hyperbaric conditions, our results at least appear consonant with those cited by Alkana and Syapin.

Several studies of infrahuman mammals, on the other hand, apparently contradict our findings. For example, when mice were anesthetized with large injections of ethanol (3.2 g/kg body weight) and subjected to various increases of atmospheric pressure and oxygen mixtures, "treatment with 1 or 2 ATA 100% oxygen, 1, 2, or 4 ATA 20% oxygen-80% helium, or 2 or 4 ATA air, did not significantly change...blood ethanol concentrations when compared to 1 ATA air controls" (Alkana and Malcolm, 1979). At even higher levels of atmospheric pressure (6 and 8 ATA), BAC was in fact higher than in the 1 ATA control groups (Alkana and Malcolm, 1978, 1979). Several methodologic differences, as well as species-specific metabolic processes, must be taken into account before any conclusions can be suggested, even though Alkana and his colleagues consistently report that hyperbaric environments of specific types do antagonize ethanol depression without accelerating ethanol metabolism.

BAC data collected by Mallach et al. (1972), whose pardigm provided us a model for alcohol dosage and ingestion rates, are available for comparison with our BAC results. After fasting, their nonalcoholic subjects consumed 0.75 mg alcohol/kg body weight within ten minutes (versus fifteen minutes for our subjects). At seven points in their 370-minute record and our two 260-minute records, equivalent time intervals are available for comparison of BACs (see Table 3). At all points but the first,[7] their normal control subjects and ours appear to have similar BACs, but throughout the records our two experimental conditions (hyperoxyc and normoxyc gases under hyperbaric pressures) show a more rapidly declining BAC. We must tentatively conclude, therefore, that 2 ATA pressure, with or without hyperoxygenation, accelerates decline of the BAC curve in nonalcoholic human subjects, in support of our first hypothesis.

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insert Table 3 here

Results of the cognitive and motor performance tests are ambiguous and equivocal at best. In some cases subjects performed better when intoxicated than when sober, in other cases hyperbaric oxygenation offered no advantages over the other conditions, and in some cases hyperbaric oxygenation was marginally effective in altering performance. Specifically, performance on Digit Span forward ($p < .10 > .05$), and performance on the first trial of the serial-learning task ($p < .01$) were

the only dependent variables that improved when this principal experimental condition was in effect. Also, when subjects were sober, hyperbaric oxygenation appeared to enhance some performance variables over other sober conditions, but not consistently. Therefore, our second and third hypotheses are not supported.

Several alternative explanations of these results are possible. As Schmitz (1977) has pointed out, many of the studies reporting positive results (as well as those reporting no significant effects) were seriously flawed in methodology, diagnoses of "senility" were often confused with those of "cerebral arteriosclerosis," cultural differences in attitudes toward learning and test-taking were not controlled, subjects undergoing hyperbaric oxygen therapy were treated differently by staff members, some of the tests used to measure deficits were of questionable validity, and other sources of variance were introduced. It is possible, therefore, that one of our basic premises may have been incorrect and the efficacy of hyperbaric oxygen therapy has been exaggerated. That this may be the case has been stated quite strongly: "...it behooves all scientists, researchers, physicians, and health professionals to state clearly that there is no sound, validated, scientific basis to substantiate claims that hyperbaric oxygenation is of benefit in treating the central nervous system effects of aging" (Schmitz, 1977, p. 339).

It is equally possible that the acute effects of intoxication

studied in this project are in no way comparable to the acute and chronic effects of alcohol studied by Soviet hyperbaric scientists (Tishchenko, 1976; Spivak, 1977) or the chronic effects of aging studied by the American group (Jacobs *et al.*, 1969). And certainly our subjects' relatively brief and infrequent exposure to the hyperbaric oxygenation procedure is in no way comparable to those exposures used by the other researchers. Perhaps the abilities and functioning we chose to investigate (rote verbal learning, nonverbal symbolic learning, motor control and effects of fatigue, short-term memory and attention/concentration) are differentially sensitive to the acute and chronic insults of ethanol, and our repeated-measures design may have permitted learning to take place, sufficient to obfuscate any deficits (even though equivalent forms for retesting were available for most measures). Relatedly, it is possible that subjects were not sufficiently intoxicated for those measures to detect any functional impairment. Our BAC data indicate that although subjects were indeed intoxicated under the definition of Wisconsin statutes, by the time performance testing had begun alcohol levels had declined to 0.080-0.088 in the baseline condition, and 0.074-0.077 in the hyperbaric conditions. Future studies of this sort should ensure adequately high doses of ethanol such that subjects are in fact intoxicated at the time of testing, now that our data suggest a "hyperbaric BAC curve."

We are satisfied that this study has fulfilled some of the

expectations of a small pilot project and would encourage further research along these lines to determine the efficacy of hyperbaric oxygenation in the treatment of acute and chronic alcohol intoxication.

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Notes

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[2]Subjects later reported that the placebo and alcoholic beverages were virtually indistinguishable by taste at the time they began drinking.

[3]Subjects were not told, however, when they would be receiving placebo or alcoholic beverages, nor were they informed of when they would be breathing oxygen or oxygen and nitrogen.

[4]Subjects entered the air lock (the forward portion of the chamber), which was then gradually pressurized to 2 ATA under the supervision of a technician. When the appropriate pressure was reached, usually within 4-6 minutes, the forward and aft portions of the chamber had been equalized and the hatch between the two could be opened. The technician then seated the subject in the aft area, fitted him with his mask, closed the hatch, and the forward section was then depressurized and made ready to receive the next subject. This procedure, and all others involving the chamber, was supervised by a technician within the chamber and, via closed-circuit television and intercome, by technicians and operators at the main control panel outside the chamber. All chamber procedures were constantly monitored and supervised by a physician (E.P.K.).

[5]Because subjects responded to digit span and serial-learning tasks orally, a different form of each test was used for each subject during each session in the chamber, thereby precluding

the possibility of subjects learning from each other or from previous exposure to materials.

[6]All reported p values are one-tailed.

[7]Interestingly, the ascending limb of the BAC for our subjects rose so rapidly that by the time we took our first measurement (fifteen minutes after ingestion) it had already begun its descent. This phenomenon is quite common, as explained by Haggard, Greenberg, and Lolli (1940) in terms of an "overshooting [effect that] results from a rate of absorption which not only exceeds that of oxidation and elimination, but also distribution" (p. 686).

Table 1. Experimental conditions and schedule of sessions.

gases breathed	Beverages Served: Alcohol	Beverages Served: Placebo
Oxygen	Sessions 2, 4	Sessions 5, 8
Oxygen + Nitrogen	Sessions 3, 7	Sessions 1, 6

Table 2. Means and standard deviations for performance measures under experimental and control conditions.

Sessions	ML alc	BAC-1	BAC-2	BAC-3	FO-1	FO-r	DSp-f	DSp-b	DSp-t	DSy	SL-1	SL-2	SL-3	SL-4	SL-5	SL-6	SL-7	SL-8	SL-9	SL-10
2 + 4	153.00	.102	.082	.058	48.84	54.56	8.00	6.00	14.00	48.50	20.83	21.88	21.86	21.88	34.38	25.00	56.01	60.77	60.75	63.75
	23.38	.036	.031	.022	4.88	8.48	1.05	1.69	2.71	13.57	19.09	18.75	21.35	15.73	27.72	35.36	28.01	27.24	30.59	24.60
3 + 7	149.00	.099	.094	.057	46.51	55.32	7.75	5.25	13.00	51.75	00.00	20.83	14.58	25.00	33.33	46.88	50.00	62.50	68.75	62.50
	23.12	.037	.013	.012	6.07	7.03	1.36	1.36	2.29	16.39	00.00	15.14	12.29	17.68	21.89	27.72	43.30	35.36	26.52	35.36
5 + 8					50.98	58.97	7.92	6.58	14.50	55.67	8.33	30.21	45.83	55.21	60.42	75.00	75.00	62.50	62.50	75.00
					5.35	7.89	1.24	1.31	1.98	16.49	13.41	15.50	21.54	25.82	27.09	22.54	23.15	27.00	30.62	12.50
1 + 6					49.57	57.49	7.64	6.00	13.64	50.09	0.00	13.89	27.78	47.22	47.22	51.39	54.69	57.81	53.57	56.25
					4.22	6.93	1.12	1.48	2.01	16.07	0.00	7.51	8.33	17.43	26.35	25.35	24.03	30.57	33.63	31.38
C					49.47	57.76	6.78	6.33	13.78	49.33	7.81%	23.61%	27.78%	52.78%	59.67%	62.50%	55.36%	66.07%	71.43%	75.00%
					3.50	5.95	2.54	1.23	1.92	16.29	9.30%	19.21%	16.27%	30.48%	32.89%	25.00%	26.87%	22.49%	21.30%	15.81%
9	148.67	.119	.079	.064	52.09	61.27	6.83	6.33	13.17	57.67	0.00	29.17	43.75	52.08	56.25	54.17	45.00	55.00	47.50	50.00
	27.95	.013	.017	.018	9.29	6.52	1.47	0.82	1.60	14.19	0.00	20.41	32.36	24.26	31.37	30.28	22.71	20.92	40.89	27.95

ML alc = mean dose, ml 100 proof beverage.

BAC-1, -2, -3 = mean BAC (mg%/dl) at 15 min. after ingestion, 60 min. later (at time of testing), and 60 min. later (at exit from chamber), respectively.

FO-1, -r = mean finger oscillations per 10-sec. trial for left and right hands, respectively.

DSp-F, -b, -t = mean number correct responses, Digit Span-forward, -backward, -total, respectively.

DSy = mean number correct responses, Digit Symbol.

SL-1...SL-10 = mean % correct responses, serial-learning task, trials 1-10, respectively.

For all entries, means are shown in the upper line, standard deviations in the lower line.

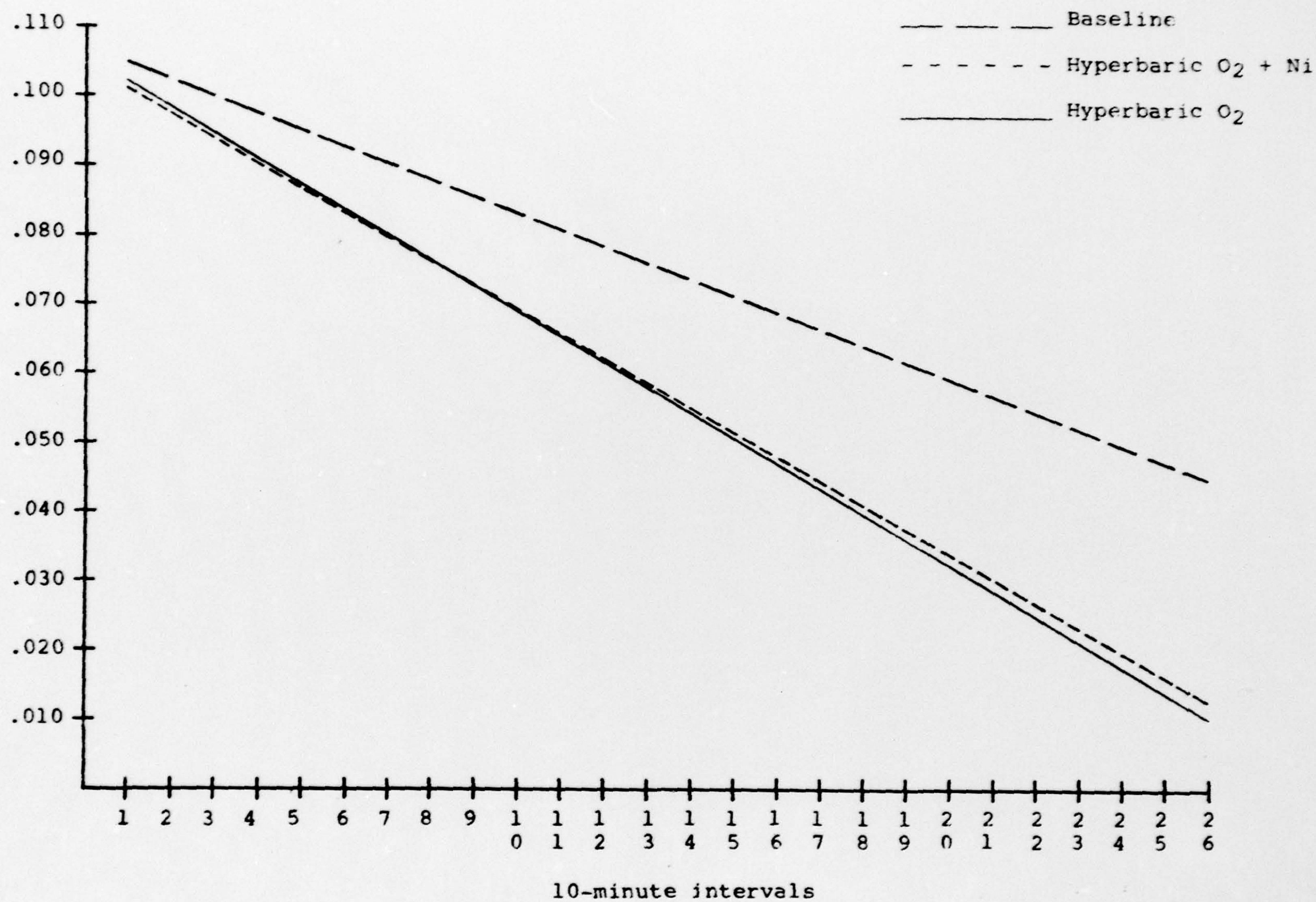

Figure 1. Four-hour BAC curves (interpolated through regression) beginning 10 minutes after last drink.

Table 3. Mean BAC values (mg %/dl) for three groups of subjects.

minutes since last drink	Mallach *et al*.	normobaric/ normoxyc	hyperbaric/ hyperoxyc
30	.070	.0997	.0945
50	.092	.095	.0872
70	.093	.0902	.0798
100	.087	.0831	.0688
130	.077	.076	.0578
190	.065	.0618	.0358
250	.051	.0475	.0138

Abstract

To determine the efficacy of hyperbaric oxygenation in reducing the cognitive and motor performance deficits usually associated with acute alcohol intoxication a 2 x 2 repeated-measures experimental design was used, in which 6 nonalcoholic adult male social drinkers ingested either 0.75 mg ethanol/kg body weight, or an equivalent amount of placebo beverage, and then spent 2 hours in a hyperbaric chamber at 2 ATA, where they sat quietly and breathed either 100% oxygen or 11.5% oxygen-87.5% nitrogen. Throughout the procedures Breathalyzer readings, and venous and arterial blood samples were taken periodically, and BAC and blood gas analyses were conducted. After the first hour, cognitive and motor performance tests were administered for a 15-minute period, during which subjects breathed chamber air for approximately 9 minutes, followed by another 50 minutes of sitting quietly and continuing to breathe via individual masks. Hyperbaric procedures in general, but not the 100% oxygen condition specifically, appeared to accelerate the decline of BAC. Results of testing were equivocal but hyperbaric oxygen procedures appeared to have no general effects on performance. Alternative explanations for the observed effects, as well as for the hypothesized effects which did not obtain, are provided, and suggested directions for future research are offered.